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Role of intra compartment testing in chronic exertional compartment syndrome: An uncommon presentation of a common syndrome

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Abstract
A classic clinical presentation for chronic exertional compartment syndrome (CECS) in an active military service member with exertional pain in the lower legs after increased physical activity but with paradoxical or equivocal intra compartment pressure (ICP) testing. Patient's symptoms were improved with empiric standard of care treatment for chronic exertional compartment syndrome. This brings to attention, the validity of the CECS gold standard modified Pedowitz diagnostic criteria and how it is tested.

Keywords: Chronic exertional compartment syndrome, Pedowitz criteria, intra compartmental testing, lower extremity

Introduction
Chronic Exertional Compartment Syndrome (CECS) is a relatively common diagnosis in young active athletes and in the military (Callender). The condition most often affects the anterior and deep posterior compartments with increased compartment pressure that can cause pain, weakness, and paresthesia. (Martens). CECS is typically diagnosed with intra compartment pressure (ICP) testing via the modified Pedowitz criteria: a resting pressure measurement ≥15 mm Hg, and/or a measurement taken 1 minute after exercise ≥30 mm Hg, and/or a measurement taken 5 minutes after exercise ≥20 mm Hg (Fraipoint). The patient presented with classic symptoms of Chronic Exertional Compartment Syndrome (CECS).

Case Presentation
Patient is a 24-year-old male solider who developed bilateral leg pressure and shin pain up to the myotendinous junction of the peroneal muscles during long distances runs and road marching. The pain was accompanied by numbness and tingling that began distally in the toes and spread dorsally and laterally from the feet to the ankles. Additionally, reported foot drop and muscle herniations in the lateral compartments with exercise. He also experienced redness of the feet with hot showers. The symptoms started and was worse in the right leg and then developed in the left leg with continued activity. After discontinuing the provoking activities, the symptoms resolved within a few minutes. He did not have any of these symptoms while playing sports in high school but only ran sprint distances during track and football.
He underwent ICP testing with diagnostic resting pressures of the right anterior (16 mm Hg) and bilateral lateral compartments (R: 23 mm Hg, L: 25 mm Hg) but paradoxical findings with decreased pressures in these compartments after activity per table 1. Repeat ICP testing a week later with a different provider showed only a mildly diagnostic finding at rest in the less symptomatic left anterior compartment (15 mm Hg) but none of the exertional pressures met diagnostic criteria. Specific timings of the post-exercise measurements were not documented. The differential for the patient included superficial peroneal nerve entrapment and popliteal artery entrapment syndrome. Superficial peroneal nerve entrapment could cause symptoms similar to the presentation but did not fully match the patient's pain and paresthesia locations. Popliteal artery syndrome was not wholly consistent with patient's presentation since it typically presents with a claudication symptomatology with aching pain in the calf muscles rather than numbness in the feet. Despite the equivocal and paradoxical

findings, the patient underwent empiric treatment of physical therapy and Pose method of running for CECS. The patient developed improvement of symptoms with running but had not returned to road marching yet. Patient was recommended to continue trial of running style modification, dry needling, and trigger point injections with consideration of compartment pressure re-testing if unsatisfactory.

Table 1: Compartment Testing

	Right LE		Left LE	
Aug 23	**Resting**	**After Exercise**	**Resting**	**After Exercise**
Anterior	16	7	7	14
Lateral	23	18	25	17
Superficial Posterior	8	14	9	10
Deep Posterior	13	18	11	20
Aug 31				
Anterior	13	15	15	19
Lateral	10	18	10	22
Superficial Posterior	07	08	10	08
Deep Posterior	11	11	10	12

Discussions

The pathophysiology of CECS is unclear but likely is multifactorial. Per Schubert's CECS review, theories included increased fascial stiffness/thickness, muscle microtrauma causing delayed reoxygenation and potentially ischemia, inflammation increasing fluid flow with subsequent increased volume and pressure, and increased pressure as a source of increased pain receptor stimulation. 10-60% of CECS patients also had small fascial defects in the lower leg.

When CECS patients develop increased compartment pressure from exertional activity, they can then develop pain, paresthesia, and muscle weakness. A patient can meet the modified Pedowitz criteria for elevated resting or post-exertional pressure readings; however, what does the elevated resting pressure indicate if the patient doesn't have any symptoms at rest? A potential thought is that an acute increase in physical exertion, causes muscular microtraumas and the body's inflammatory healing process causes myofascial adhesion which stiffens/thickens the fascia reducing compartment compliance during exertion. This reduced compliance may then cause a positive feedback loop of delayed reoxygenation unable to meet exertional oxygen demands, ischemia, inflammation, and increased fluid in a compartment with decreased compliance further increasing pressure. This reduced compartment compliance may also contribute to the association of fascial defects and muscle hernias. However, it would be expected for a patient with elevated resting pressures to also have elevated exertional pressures, which was not present during the patient's first ICP measurement.

The initial ICP testing was diagnostic at rest of the right anterior compartment and the bilateral lateral compartments when the patient was not experiencing any symptoms and then paradoxically decreased after exertion with reproduction of symptoms. There is a possibility the test was not performed or documented correctly so a repeat test was done to confirm findings. However, the repeat test a week later was not diagnostic in any of the compartments other than the left anterior compartment, which was not associated with any symptoms at rest and was the leg with less severe symptoms. Factors affecting ICP accuracy can include proper equipment use, correct anatomic placement of catheter tip, needle insertion depth and angle, and position of extremity during measurement. The patient's equivocal ICP findings with classic symptoms of CECS should prompt the question; is there a low pressure subtype of CECS?

There is no standardized ICP test protocol for diagnosing CECS which causes large variability of ICP findings and diagnostic thresholds. Per Aweid's review of ICP testing, pre-exercise mean values for CECS patients ranged from 7.4 to 50.8 mm Hg, and 5.7 to 12 mm Hg in controls. Measurements during exercise showed mean pressure readings ranging from 42 to 150 mm Hg in patients and 28 to 141 mm Hg in controls. There was no overlap of pressure measurements between CECS subjects and controls in mean ICP measurements at the 1-minute postexercise timing interval with CECS pressures of 34 to 55.4 mm Hg and 9 to 19 mm Hg in controls (highest value in control was 27.5 mm Hg). Given these findings, diagnosis of CECS should emphasize on a good clinical history since ICP test findings can vary drastically. Furthermore, the degree of pressure elevation did not correlate with symptomatology or predict surgical fasciotomy outcomes (Schubert). Although this study confirms the specificity of the one-minute post-exercise; further evaluation of specific diagnostic thresholds may be warranted.

Per Lindorsson, there are significantly lower median one-minute post exercise ICP values in the superficial posterior (35 mm Hg), deep posterior (33 mm Hg), and lateral compartments (40 mm Hg) compared to the anterior compartments (47 mm Hg). The modified Pedowitz post-exercise diagnostic pressure criteria may not be sufficient for diagnosis of each compartment and may require further stratification per compartment. A lower threshold of the lateral and both posterior compartments may improve diagnosis and treatment of suspected CECS patients.

In addition to compartment pressure testing are there other objective markers useful in diagnosing CECS? If ischemia is associated with CECS, the levels of creatine kinase may be elevated more than the average individual without CECS. Per Kindermann, the majority of competitive athletes have elevated creatinine kinase levels; however, there is a wide range of variability of creatinine kinase levels post-exertion. Further stratification of estimated creatine kinase elevations after exercise compared may provide additional data to consider. However, some limitations include confounders such as hydration status and patient's baseline fitness level. If fascial damage and reduced fascial compliance is associated, further research on assessing fascial changes in CECS patients can be useful. Ultrasound has been used to

assess for fascial densification and may be a useful tool in developing a noninvasive diagnostic tool for CECS (Hughes).

Conclusion

The pathophysiology of CECS is uncertain but it may be related to myofascial adhesion and decreased compartment compliance that is unable to adapt to increased pressures during exertion. There is wide variability of ICP findings in CECS patients so an emphasis on good clinical history is important in the assessment of these patients. The CECS diagnostic criteria would likely benefit from further testing to achieve a standardized agreed ICP measuring protocol with an emphasis on one-minute post-exercise pressure measurements and compartment specific pressure thresholds. Further evaluation and agreement on existing CECS evaluation and novel evaluation methods would likely optimize treatment for CECS moving forward.

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